the anterior chamber. At length, after intense and protracted suffering, the swelling bursts in one or both these situations, and a mass of yellow tenacious lymph is pushed forwards, but not discharged, neither does the eyeball collapse. This lymph comes away in flakes and strings, is detached very slowly, and in proportion as it escape the pain abates, but the eye falls down within the socket, and not only is vision lost, but a very unsightly deformity remains that can only be palliated by the closure of the lids, or the adaptation of an artificial eye. It is well for the patient that this disease never attacks both eyes at the same time, and seldom passes from one eye to the other, and thus although condemned to lose the one by a painful, harassing, and nearly incurable affection, he is in less danger of total blindness than from the disease previously described—that which is usually called iritis."

52. *New Mode of Curing Ectropion.*—Dr. Blanbery regards this affection as mainly induced by a peculiar state of the orbicularis muscle, consisting in a preponderance of contractility in the fibres which surround those which form the ciliary portion of the muscle. In a man, aged fifty, with ectropion, Dr. Blanbery found a furrow below the ciliary margin of the lower lid, which became deeper every time the patient closed his eyes forcibly. He made a vertical incision in the lid, and divided some contracted fibres of the muscle, and thus removed the deformity.—*Prov. Med. and Surg. Journ.*, 22d Sept., from *Gazetta di Milano*, 27th Feb., 1847.

53. *Malignant Disease of the Eyeball.*—Mr. Henseley related to the Bath Pathological Society, April 15th, 1847, the following case:—

"A female servant, twenty years of age, had been for two or three months previous to September, 1846, complaining of weakness and loss of health. She had observed a tumour above the left eye, about the size of a small marble, at the upper and inner part of the orbit. It had, on examination, much the feeling of an encysted tumour. The eye was very prominent, and thrust a little outwards. The sight at this time was not much impaired. For some time the tumour appeared stationary, but early in October it began to enlarge; the skin covering it assumed a livid hue; there was effusion beneath the conjunctiva, which produced chemosis. The eye became more prominent; she suffered great pain in the ball itself, but more in the back part of the head. The tumour continued to increase in size; the cornea separated, and the lens and humours of the eye escaped. In December, the other eye became affected, and rapidly increased in size; the cornea separated, and the humours escaped as before. During this time her strength had greatly diminished; she was scarcely able to move in her bed; the pain was most intense, coming on in paroxysms. She often fell into a comatose condition, in which she sometimes remained for twenty-four hours, and was several times thought to be dying, when she would rouse up, call for food, and drink, and make a good dinner, and then in a little time, fall into the same comatose condition again. This state continued for about six months, the intervals of consciousness becoming longer towards the close of that period. There was frequently a discharge of blood from the left eye, and a fœtid discharge from the nostrils. She died at the end of January.

On removing the scalp after death, a bluish-looking spot, about one-third of an inch in length, and one-eighth in breadth, was seen at the posterior inferior angle of the right parietal bone. On separating the pericranium, which was easily accomplished, this spot was found to be a hole in the bone, perforating it completely. Around the hole, the bone was found to be more vascular than usual, and there were several smaller perforations. The bones of the head, generally, were very thin; and on holding the skull-cap between you and the light, it appeared translucent, and at many points perforated. The whole of the inner surface, more especially the posterior portion, (the part where she had felt so much pain during life,) was rough, having the feeling and appearance of grains of sand scattered over it. At the spot corresponding to the perforation of the parietal bone, there were several very large Pacchionian glands, and the dura mater was firmly adherent to the bone, and very vascular. There was slight sub-arachnoid effusion. The anterior lobes of the cerebrum were softened and disorganized, presenting a pulpy mass, which could with difficulty be removed from its position. The dura mater around was nearly one-quarter of an inch in thickness, and semi-cartilaginous, and adhered so closely to the bone, as to render its detachment impossible A considerable portion of the orbital plates of the frontal bone, more particularly on the left side, and also parts of the ethmoid and sphenoid bones, were destroyed. The optic nerves appeared healthy; the pituitary body was much softened.—*Prov. Med. and Surg. Journ.*, Nov. 17th, 1847.

MIDWIFERY.

54. *On the Primary cause of Puerperal Fever.* By Dr. Scanzoni, of Prague.—Dr. S. commences by stating that the wound produced on the internal surface of the uterus by the detachment of the placenta during parturition, is universally regarded as the peculiar cause of puerperal fever. He considers this view too exclusive, and in opposition to it mentions two or three cases where the symptoms of puerperal fever had shown themselves before labour had commenced, and continued after it to a fatal termination, and there was no hemorrhage, nor putrefaction of the placenta to indicate that that organ had been detached before the proper period of labour. Dr. S. then believes that "the seed of the disease is sown before the commencement of labour;" accordingly he seeks for the germ of the disease in the blood, and, as the peculiar condition of the blood of a puerperal is formed from that of a pregnant female, he conceives that the special predisposing causes of puerperal fever exist in the composition of the blood. In proof of this, the author mentions that in those cases which came under his own observation, where that composition of the blood, which is peculiar to pregnant women, was totally prevented from taking place, or was modified by the presence of some other dyscrasia, the patients were never attacked with puerperal fever, although exposed, in an eminent degree, to that combination of circumstances which is supposed to induce it; and, on the other hand, when, during pregnancy, females are attacked with any disease depending on hyperinosis of the blood, such patients are very liable to suffer from puerperal inflammations, accompanied by copious fibrinous exudations. The author refers to the writings of Helm, Kiwisch, Rokitansky, and Engel, showing that the blood of puerperal females is in a state of hyperinosis, *i. e.* containing an excess of fibrine. The author saw that strong healthy women, in whom this state of hyperinosis of the blood natural to pregnancy had attained its full development, suffered, during a severe epidemic, under that form of puerperal disease which gave indications of an excess of fibrine in the blood, viz. endometritis or peritonitis, accompanied with copious fibrinous exudation, such as is met with in pleuritis and pneumonia.

Physiologists are as yet unable to explain how this augmentation of fibrine is produced. Fibrinous deposits are frequently found in or upon the placentas of mature children, but very rarely has the same alteration been found in the case of premature fœtuses. These deposits are mostly confined to the two last months of pregnancy, and their existence may, according to our author, be accounted for by the active interchange of material which, toward the end of pregnancy, takes place between the maternal and the fœtal blood; the former abounds in fibrine, and if the consumption of this element on the part of the child be diminished, whilst the supply afforded by the mother remain the same, fibrinous deposits must take place, and these probably on the inner surface of the uterus: this is a fact our author has been unable to ascertain, as the inspection of the bodies of women dying during pregnancy are exceedingly rare. The fibrinous deposits in the placenta, however, according to Dr. S., prove that the fœtus does not consume all the fibrine supplied to it, and they lend probability to the idea, that in consequence of the continually increasing supply of fibrine in the circulation of the mother, and the comparatively small consumption of that element on the part of the child, an abnormal accumulation of fibrine must be the result, constituting hyperinosis in the mother.

This fibrinous diathesis, in pregnant females, may pass into secondary dropsy or purulent deposit; secondary dropsy follows from the anæmia gradually devel-